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Antibiotics

THE forthcoming release (on November 1st) of both aureomycin and terramycin for general use by veterinary surgeons in Great Britain would appear to be an appropriate moment for a brief consideration of the history, and of the field of use, of some of the better known antibiotics.

The story of penicillin and its accidental discovery by Sir Andrew Fleming has been told many times, because the production of this drug opened up a completely new field of research and investigation into the treatment of bacterial infections. The idea of using the product of mould growth for the inhibition of bacterial multiplication stimulated scientists all over the world to search for other moulds which might have an even wider action than penicillin. It is not necessary to discuss the merits of penicillin as we have all had an opportunity to test its efficiency in the treatment of the many disease problems of the farm animal, and it would not be an exaggeration to say that this drug has altered the whole concept of the treatment of bacterial infections, especially those of the udder.

Penicillin was followed in 1944 by streptomycin which was produced from the group of micro-organisms known as the "actinomycetes," and this drug in addition to being effective against some gram-negative organisms showed great promise in the treatment of *Mycobacterium tuberculosus*.

In 1947 a group of workers in the research laboratories of Parke Davis & Co. reported the discovery of a hitherto unknown species of Streptomyces which had been isolated from a sample of soil in a field in Venezuela. The organism was closely related to *Streptomyces griseus* (the source of streptomycin) and it was named *Streptomyces venezuelae*. The antibiotic which was isolated was named chloramphenicol and it was unusual in that it was a derivative both of nitrobenzene and dichloroacetic acid, a substance not previously found in a natural product. As soon as its structure had been identified, it was synthesised and it became the first antibiotic to be produced other than from a mould growth. Chloramphenicol was found to be effective when given orally against gram-negative organisms as well as rickettsia and some viruses.

Following the isolation of streptomycin and chloramphenicol, investigation continued into the production of antibiotics from the actinomycetes and in 1948 Duggar, working in the Lederle Laboratories Division of the American Cyanamid Company, announced the discovery of an antibiotic from a new species of this group of organisms. The

organism was called *Streptomyces aureofaciens* and the antibiotic was called aureomycin. Since that date the drug has been given the chemical name of chlortetracycline, although it cannot be produced synthetically. Aureomycin was found to be effective when given orally against numerous gram-negative and gram-positive organisms, also against rickettsia and viral infections, and it also had a low level of toxicity.

In 1950, Finlay and co-workers, of Chas. Pfizer and Company, isolated *Streptomyces rimosus*, whose active principle was given the name terramycin and is now known chemically as oxytetracycline. Terramycin is closely related to aureomycin in structure and thus has a similar wide spectrum of activity.

The release of these new antibiotics with their wide spectrum of activity should be of great value to the clinician but, at the same time, it will emphasize the value of laboratory facilities for bacterial examinations and also the importance of the differential diagnosis of such conditions as mastitis and the pneumonia complex. In the medical field it is a common practice to test organisms for their reaction to the various antibiotics before treatment is advised ; such a procedure will not always be practicable in the veterinary field, but it is important to realise that the value of the newer antibiotics is enhanced if they are used under the most effective conditions.

Aureomycin and terramycin are both stable in dry form and soluble in water, and they can be given intravenously, orally, and by intramammary injection. Because of the effect of an antibiotic on the ruminal bacteria, antibiotics should not be given orally to adult ruminants. It is usual to group aureomycin, terramycin and chloramphenicol as " broad spectrum " antibiotics because of their activity against gram-positive, gram-negative, rickettsia, large viral and protozoan organisms.

Because of their wide range of activity, they are of particular value in the treatment of non-specific clinical conditions. As we all know, such conditions occur as frequently in veterinary as in medical practice, and it is because therapy of this kind requires clinical observations, that these drugs have been so carefully kept in professional hands.

Both aureomycin and terramycin have been available to veterinary surgeons in America for the last four years, and there is a considerable literature on the use of these drugs on both large and small animals. Veterinary surgeons in this country, therefore, will now have the opportunity of comparing their results with those of their American colleagues.

Pan-African Zootechny Congress, Algiers

A most successful technical conference took place in the picturesque town of Algiers from October 17th to the 23rd. This conference, which was organised by the Société Vétérinaire de Zootechnie d'Algérie, was given the name of " Première Journées Panafricaines de Zootechnie." The programme was arranged to appeal particularly to those who were professionally concerned with the development and improvement of animal products in the African continent. The President of the Organising Committee (Dr. Jore d'Arces) explained in his opening address that his Society had hoped to bring together for discussion and personal contact a large number of veterinarians and others, with the principal object of seeking ways and means to increase the amount of milk and meat for the 200 million people who live on the African continent. Although the main object of the conference was to discuss zootechnical subjects, it was impossible to divorce pathology from the programme. The full development of the animal resources of the African continent must depend not only upon improvement of animal husbandry methods, but also, of course, on the control of disease.

The conference presented a nice balance of strictly zootechnical discussions and discussions on animal disease control. It was very well attended, and great interest was maintained in the series of papers, and the discussions which followed them. It seems most unfortunate that this conference, which was organised by our French colleagues in Algeria, should have received such poor support from British people. One would have thought that the subjects discussed were of vital interest to all African Colonial territories, and also to the territories of the Union of South Africa, and the Central African territories. This was a first-class opportunity of attempting to achieve co-operation between the French-speaking and the English-speaking veterinary departments in the African continent, yet we found present only two veterinarians from Nigeria, two from the Sudan, and one from Kenya, and apart from these there were two representatives from F.A.O., and two British veterinary surgeons attending the conference in a private capacity. The official languages of the conference were French and English, and an efficient team of translators was available, so that all the English-speaking delegates were able to follow and take part in the whole of the conference without difficulty.

The subjects of the discussions (which will be reported more fully in a later edition of THE VETERINARY RECORD) were as follows:—

Artificial Insemination (Opener—James Anderson, O.B.E., Kenya); Contagious Bovine Pleuropneumonia (Opener—Dr. Nunes da Silva Araujo, Angola); Trypanosomiasis (Opener—Dr. Guyaux, Belgian Congo); Utilisation of Meat Resources in the African Continent (Opener—Dr. Receveur, Tchad, A.E.F.); Rinderpest (Opener—Dr. Mornet, Sénégal, A.O.F.); Bovine Piroplasmoses (Opener—Dr. Donatien, Algeria); Mineral Deficiencies (Opener—Dr. Louw, South Africa).

(*Continued on page 678*)

Husk in Cattle

A REVIEW OF A YEAR'S WORK*

BY

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Introduction

THE problem of "husk" in cattle has been under investigation at this school since the summer of 1952. This paper is a report of our findings to date, and although the work is incomplete and much remains to be done it is considered that an interim report will be of some interest to those concerned with the disease. In this work we investigated outbreaks of coughing and respiratory disease, both indoor and outdoor, occurring in the summer and autumn. Most of these were in the S.W. Scotland area but additional material has been obtained from various parts of Scotland and England. In addition to this a large number of lungs were examined at a local knackery.

The outstanding problems of parasitic bronchitis may be summarised as follows :—

1. There are no figures for the incidence of parasitic bronchitis in Britain with respect to geographic distribution, morbidity and mortality.
2. Important details from the control point of view of the life-cycle of *Dictyocaulus viviparus* are unknown.
3. The effect of pasture management on the epidemiology of the disease.
4. Little is known of the relative effects of the lungworms and the lesions associated with their presence in the production of clinical signs.
5. Symptoms have never been correlated with morbid changes in the host.
6. Very little is known of the mechanism and site of immune phenomena and their significance in the field.

INCIDENCE

(*a*) Knackeryard Survey

The largest knackery in this area was visited several times weekly. Arrangements were made to examine almost all bovine lungs irrespective of the age of the animal or the cause of death. In almost all of the husk cases, the lesions were considered severe enough to be the cause of death. Unfortunately calves of under three months of age are not opened at this knackery, being processed whole for economic reasons ; this age group therefore is not included in our figures. The lungs were graded roughly into two groups, viz., stirks and adults. The results for August, September and October are tabulated in Table I.

It is reasonable to assume that the numbers of carcases and the types of disease in this knackery reflect the incidence of fatal disease in the area.

One striking feature which then emerges is that approximately 50 per cent. of all bovine deaths in this area in the period August to October, 1953, were attributable to husk, *i.e.*, it was the most important killing disease of cattle during these months.

A further point of interest is the number of adult lungs affected. Husk is commonly supposed to be a disease primarily of the first year of life, but in this series of 109 cases 46 cases, or 42 per cent., were adults. The highest number of adult cases occurred in September although the highest ratio of affected adults to stirks was in October. This survey was also carried out during the winter and spring months in order to find if adult lungworms were present in bovine lungs during this period. During the spring months of 1953 a few cases of bovine lungs containing adult lungworms were found ; this year arrangements have been made to enlarge this survey due to the improved co-operation and enlargement of the knackery. In February and March, 1954, 211 pairs of stirk and adult lungs were examined of which 11 (5 per cent.) contained small numbers of lungworms (*i.e.*, up to 30) in their bronchi. It is hoped from this extended survey to obtain some information on the carrier problem in both adults and stirks.

(*b*) Outbreaks Investigated ; Technique of Farm Examination

This year we visited 40 farms ; in nearly all cases these were visited on more than one occasion. From 30 of these we obtained at least one animal for autopsy and as many as five on several occasions. Another five of these outbreaks were confirmed as being parasitic bronchitis by faecal examination.

Repeated routine visits are being made to these farms in order to study the recovery of affected animals, their possible significance as carriers and the effect of pasture management. Routine faecal examinations are being carried out on these herds and the pattern of pneumonia in the indoor stock studied.

The investigation of each outbreak was conducted as follows. A clinical examination was made of each affected animal and this was followed by an examination of a representative sample of so-called normal animals of all age groups from newborn calves to adult cows. The clinical procedures which we found to be most useful were counting the respiratory rate and auscultation of the chest by a phonendoscope of large (2-inch)

* This work was done with the aid of a grant from the Agricultural Research Council and is the substance of three papers presented to the S.E. Division B.V.A. at Maidstone on October 23rd, 1953 ; results obtained since then have been added to bring the subject matter up to date. The paper was received for publication in April, 1954.

TABLE 1

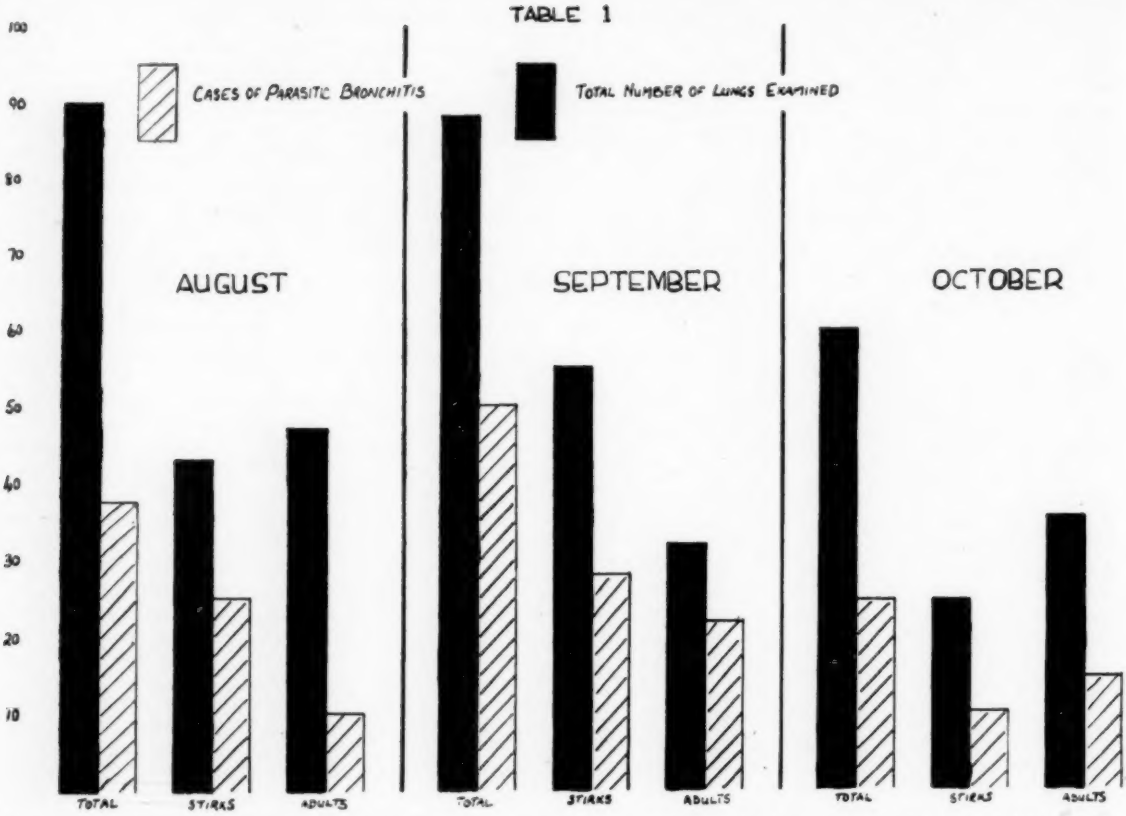

TABLE 1.—The incidence of parasitic bronchitis in lungs examined at a knackery in August, September and October, 1953.

diameter ; this instrument is of much greater value for use on the bovine than the stethoscope. In the normal bovine standing at rest in a cool calf house or byre we found the respiratory rate to be under 20 per minute. It increased with an increase in temperature and/or humidity to as much as 40 per minute in dairy stock and 70 in beef breeds. We found it necessary to examine several or all of the animals in a group before assessing the significance of the respiratory rate in hot or humid conditions. At this low normal respiratory rate we found that the only sound to be heard on auscultation was a short, soft vesicular one at the end of inspiration, it being slightly harsher in the adult bovine. This sound was often difficult to hear on each inspiration particularly in the fat animal. Sniffing on the part of the animal, particularly the calf, was found to exaggerate this sound to an abnormal degree and had to be guarded against. Faecal samples were taken from all of the selected animals. As complete a history as possible was obtained from the farmer of his system of husbandry with particular regard to pasture management. It was often found necessary to ask the same question in several different ways in order to get accurate data, *e.g.*, some farmers do not consider their young stock to be "out" at grass unless they are outside both day and night ; or cattle may have had regular transit over a field although to the farmer it had not been "grazed."

A number of animals moderately affected with parasitic bronchitis were purchased to study as potential carriers during the following spring.

OUTDOOR HUSK

Epidemiology

The way in which lungworm infection persists from year to year on a farm has never been satisfactorily elucidated. Three possibilities have been suggested.

1. The larvae survive on the pasture over the winter.
2. Adult lungworms persist in the bronchi at least until the following spring.
3. Infective larvae ingested by a relatively immune animal undergo an inhibition of development which may prolong the prepatent period until the following spring.

1. *Survival on Pasture*

The longevity of the infective larvae of *D. viviparus* on herbage has been a matter of some controversy. On dry pasture in America, Porter (1942) stated that the larvae lived for one week but not for six or seven weeks. In Germany, Wetzel (1945) concluded that the larvae do not survive the winter on pasture. Taylor (1951) on the basis of field investigations suggested that the larvae can survive for almost 12 months although in a review of recent work at Weybridge, Stableforth (1953)

said that the larvae survived for only a month in spring and summer and up to three months at other times.

In order to determine if larvae may survive the winter under the field conditions obtaining in the West of Scotland, three plots of ground were fenced off and heavily contaminated with infected faeces. Two of these plots were grazed with young susceptible calves after six months and the third plot after 13 months had elapsed. Animals from each of these groups were found to be infected with lungworms at autopsy. This shows that under the field conditions obtaining in this area during 1952–53 the larvae of *D. viviparus* survived at least 13 months. These experiments are being repeated on a larger scale this year.

In addition to these pasture experiments, samples of heavily infected faeces have been placed on grass grown in small pots and kept at atmospheric conditions of temperature and humidity (Bell & Urquhart—unpublished). The grass 3, 6, 9 and 12 months after infection is being fed to calves reared in isolation using only sawdust as bedding and fed on milk and autoclaved hay. To date results are available from the three-, six- and nine-months old samples, all of which produced successful lungworm infections. It is of interest that the six- and nine-month-old grass plots were exposed to severe frost during the winter months.

2. *The Possible Significance of Carriers*

Naturally infected cases of lungworm disease which were purchased last year from farms are being studied at the veterinary hospital in order to determine if they play any part in spreading the disease during the following year. This work is being backed up by repeated visits to farms where known cases of parasitic bronchitis are housed. The results of this work are not yet available but two observations made so far are of interest. In a preliminary investigation on the above lines in 1952–53 five heifers, all natural cases of husk, were left outside until February when they were housed. During the late autumn and winter the faeces of these animals were consistently negative for lungworm larvae but on several occasions during April and May two of these five beasts were excreting 25 to 50 larvae per gramme of faeces. We investigated an outbreak of parasitic bronchitis in Wigtownshire in December, 1953 ; in this area it is common practice to leave the young stock outdoors during the winter. Six out of 15 calves died and faecal counts of over 1,000 larvae per gramme were recorded. At the end of February, 1954, all of the survivors were in poor condition. When the faeces of these were examined one was found to contain 250 larvae per gramme and another several larvae per gramme.

Build-up of Infection

One interesting point which emphasises the ease with which the disease may be spread is the large larval counts which may be encountered in the faeces of infected animals. It is common in field cases to obtain counts ranging from 150 to 1,000 larvae per gramme : frequently the counts are in excess of this, the highest which we recorded being 7,000 larvae per gramme from a cow which died a fortnight later. These high counts may be maintained over a period of several weeks and it is thus obvious that a field may be heavily contaminated by relatively few infected animals. In our experience a single dose of 5,000 infective larvae is often sufficient to kill an experimental calf within 30 days and in the field this lethal number will often be contained in a few grammes of faeces or in the grass immediately surrounding it. Another point is that although only a small percentage of larvae survive until the following spring, their absolute numbers may be quite high.

A prominent epidemiological feature of parasitic bronchitis in calves is that the greatest number of cases occur in August and September. Most of these calves go out to grass in May and it is therefore 10 to 15 weeks before they become clinical cases. It is suggested that the calves probably contact an initial light infection in May and it is the larvae from these lungworms or even from the second generation which produce the marked clinical symptoms in the late summer and autumn. This interpretation of the "snowballing" effect of an initial small dose is purely speculative as we do not know enough about such factors as the immunological response to such a pattern of infection but in the light of present knowledge it is the most likely explanation of the autumn incidence of the disease.

Course of the Disease

The outbreaks which we visited were all, with one exception, associated with pre-grazing of the infected pasture by older bovines or by affected calves during the previous autumn. In the exception, calves were turned out on to undersown grass. Corn had been taken off this field the previous autumn, and since then the field had been kept closed until the calves were admitted. The cause of the outbreak was obscure until it was found that the stubble had been manured during the autumn.

In our cases severe symptoms did not generally arise suddenly ; the usual course was that an initially moderate tachypnoea and coughing became markedly worse over a period of several weeks. Apparent exceptions to this occurred in some outbreaks involving adult cows and heifers ; these suddenly became very ill with severe dyspnoea and several died after a short interval. The other cows in these herds on casual examination appeared normal, but on closer examination, some showed accelerated respirations and harsh bronchial breathing on auscultation. From subsequent experience with some of these animals it is probably the case that these animals which suddenly became severely ill were actually showing similar slight symptoms previously, but these had passed unobserved by the stock owner. An outbreak of this type is described below.

The present owner had occupied the farm for seven years. During that time he had not had an outbreak of husk although some of the adult cattle had coughed a little in the autumn. Some morbidity and mortality had been experienced in calves in their first month of life ; this was mainly associated with scouring.

On this farm calves born after January are not allowed out to pasture that summer. Calves from the previous year go on to a field which some years ago was undersown with grass while producing its last crop of corn ; it was then reserved for first-year calves and no older animals have since been allowed on to it.

The owner has never noticed coughing among calves on this field.

The cropping policy lays emphasis on grass and silage production with a fair amount of hay but only a comparatively small acreage of oats and roots. The whole farm has been ploughed during the past seven years.

On August 28th the cows and heifers were grazing two fields ; 39 milking cows and newly calved heifers were in one field, and 15 dry cows, in-calf heifers and bulling heifers were in another. Eighteen calves at grass for the first year were in the field already described while 12 younger calves were still in the calf house ; the ages of the latter varied from a few days to five months.

For about two weeks before August 28th the owner had noticed a considerable amount of coughing from both the lactating and non-lactating adult stock. They had been grazing the same fields for the whole of August and had been on them earlier in the summer as well. On August 23rd a number of newly calved heifers had suddenly become dyspnoeic and the veterinary surgeon had been called. The clinical condition of the animals had deteriorated and one died on the morning of the 28th.

When we first examined this herd on that evening all the adult animals were in the field. About half a dozen appeared to be seriously ill and were standing with necks outstretched and coughing badly ; some were grunting while others were tachypnoeic but still continuing to eat. The afternoon milk yield had been very low.

Autopsy of the dead animal revealed only a light infestation with lungworms but a massive consolidation of all lobes of both lungs ; there was some emphysema but little obvious oedema.

A few of the most dyspnoeic animals were driven slowly into the byre and found to have respiratory rates between 90 and 110 per minute ; most of these animals grunted on expiration. On auscultation there was harsh bronchial breathing, many rhonchi and much emphysematous crackling to be heard.

Arrangements were made to examine the whole milking herd the following morning including faecal examinations for larvae.

On the 29th, 25 of 39 milking animals were found to have respiratory rates of more than 60 per minute, 10 between 30 and 40, whilst four were in the 50s. In addition to harsh bronchial breathing which all of the 25 markedly tachypnoeic animals revealed on auscultation, 18 of them had harsh adventitious rhonchi and/or emphysematous crackling.

The remaining 15 dry cows and in-calf heifers were not examined on that day as they were in the field and considered by the owner to be normal or not seriously affected. None of the calves at grass for the first time showed any coughing or obvious tachypnoea. The young indoor calves also appeared normal and no cough could be elicited on pinching the trachea. Careful auscultation could detect no abnormality.

The three worst animals were put on to a course of aureomycin given by mouth. They and another four dyspnoeic animals were kept inside night and day. Unfortunately it was not found possible to get a hayshed into which to put these animals and they remained in the byre. The milking cows were given a concentrate ration 4 to 6 lb. in excess of their milk requirements and all dry cows and bulling heifers were given 4 lb. of concentrates daily.

It is of particular interest that larvae were detected in only three of the milking cows.

On September 1st there was some improvement in the three treated animals as evidenced by the lowering of their respiratory rate but they were still markedly dyspnoeic. Two others had become worse. The herd showed a definite improvement, there now being only 15 animals with respiratory rates over 60 per minute.

The two animals which had become worse were in a corner of a hayshed ; they were markedly dyspnoeic with respiratory rates of 80 per minute. One of these was completely anorexic and had lost a lot of weight. This animal was excreting 7,000 larvae per gramme of faeces. On being admitted to the veterinary hospital it was kept alive for a further 10 days by giving it a continuous stream of oxygen by mouth. This was arranged by inserting a wooden gag into the cow's mouth and attaching the rubber tube thereto. Several gallons of water were given daily by stomach tube. The condition of the cow gradually deteriorated and it developed an expiratory grunt. As it became weaker the respiratory rate slowed to 40 to 50 per minute and the pulse volume lessened. At autopsy of this animal there was found a massive infestation with lungworms and widespread pneumonia.

Over the next two months a gradual improvement took place and the milk yield increased again. Those animals which had been kept in during the severe attack remained in for the rest of the winter. When examined in January this herd was in good condition and producing well. Several cows had continued to cough throughout the winter and two animals which had been most severely affected still had pulmonary lesions which were recognisable on auscultation. One of them was thin. Faecal samples failed to reveal any larvae. Histological examination of the lungs from the two cows which died showed widespread epithelialisation of alveoli (q.v.).

The course of a typical severe outbreak of parasitic bronchitis in calves is illustrated by the following outbreak.

On this farm the calves had been divided into two lots, the autumn calves which had been grazing a field and the spring calves which had been on a small patch of grass of about $\frac{3}{4}$ acre in size.

The 10 autumn calves were not severely affected and had been taken into a small byre. Most of them were coughing and five of them were excreting larvae in the faeces.

The 12 spring calves had also been taken in and put into the corner stall of a dark stable. Eight of them were severely dyspnoeic. Two were grunting and protruding their tongues as they gasped for air. Respiratory rates varied from 80 to 110 per minute and 50 to 60 per minute in the four not actually dyspnoeic. On auscultation harsh bronchial breathing was present in all calves while four of them had adventitious rhonchi and emphysematous crackling. The faeces of all 12 contained large numbers of larvae, two of them over 1,000 per gramme.

The owner ignored all advice about feeding and fresh

air and the calves were allowed to stay in this dark overcrowded corner. Repeated visits were made over the next two months. Six of the calves gradually became worse and died during the following month usually after 7 to 10 days of marked dyspnoea. The other six, four of which had never been severely affected, gradually recovered.

When visited during the winter, four months later, the survivors were in rather poor condition and small for their age. Two of them had recognisable residual pneumonias. In addition the young calves which had not been outside were all suffering from pneumonia to a greater or lesser extent.

It is worth mentioning that the confinement of affected animals, young or old, in small badly ventilated boxes or stalls will of itself produce a marked deterioration in the affected animal.

On the other hand the housing of affected animals in well-ventilated boxes or sheds serves the following purposes.

1. No more infective larvae can be ingested if the bedding is changed twice weekly.

2. No more larvae can be deposited on the pasture if the faeces and bedding are used for arable land.

3. Movement of the animals is limited, thus decreasing the spread of emphysema.

4. Protection from inclement weather is obtained particularly from a combination of rain and a sudden drop in the night temperature.

5. Supervised feeding of concentrates can be undertaken.

Despite these advantages of housing affected animals, it must be remembered that for at least three weeks after being taken off the infected pasture larvae are becoming adult in the lungs and the parasites will persist for several weeks in the bronchi. If they are present in sufficient numbers they will, along with the lung damage they produce, cause a deterioration in the general condition of the animal and offset any advantages obtained from good housing and feeding. This explains the disappointment often felt by owners and veterinary surgeons when animals fail to respond to any management during the first few weeks.

CORRELATION OF SYMPTOMS AND FAECAL LARVAL COUNTS

Faecal examination of all members of an apparently infected group is of much greater value than the sampling of one or two infected animals as it is our experience that several members of an infected group may show negative counts (*i.e.*, under 12 per gramme in the routine technique we employ for field sampling) and these may be among the worst affected clinically. It is important that samples should, if at all possible, be obtained per rectum and examined within 24 hours. For routine purposes the McMaster slide is very satisfactory. If no larvae are observed in the areas enclosed by the etchings, examination should include the area outwith the etched lines. If no larvae are observed a second slide should be examined. If a few of the individual counts are over 50 per gramme it is reasonable to conclude that adult lungworms are playing a significant part. In cases where all the counts are negative or below 50 per gramme the following should be considered as possible causes of the symptoms :—

(1) The migration of immature stages. It is usual in experimental calves which are infected with a single dose of a few thousand larvae to show typical symptoms of husk as soon as 12 days after infection, although larvae do not start to appear until about 23 days after infection ; also, in one cow which was experimentally reinfected one year after a severe attack of parasitic bronchitis, husk symptoms appeared after eight days. If the history suggests that the infection has been recently acquired, a second faecal examination is valuable in two or three weeks' time to establish the diagnosis.

(2) That they are primarily due to cuffing pneumonia. We have encountered several outbreaks where severe cuffing lesions were associated with small numbers of lungworms. Two such outbreaks which occurred at the end of September are of interest.

Both affected pedigree Shorthorn herds on adjacent farms belonging to one owner. The calves were still with their dams at grass and were in excellent condition apart from persistent coughing.

On the first farm a calf of five months had been noticed to be ill and tachypnoeic 36 hours previously and had been taken inside with its dam. In previous years there had been several similar cases ; calves appeared to become suddenly ill with pneumonia and died within 48 to 72 hours.

Examination showed this calf to be markedly dyspnoeic with a respiratory rate of 70 per minute. The pulse was poor in volume and 90 per minute in rate. The temperature was 104° F. and had been 105° F. the previous day. Auscultation revealed marked bronchial breathing and numerous moist râles throughout the second half of inspiration suggestive of a degree of pulmonary oedema possibly associated with the cardiac failure. Despite a grave prognosis given to the owner this animal steadily improved while receiving oxygen and aureomycin, generously supplied by Lederle Laboratories for experimental purposes, and made a complete recovery.

No lungworm larvae were found in the samples taken from this herd.

On the second farm the only animal which had been severely affected had died but there was a great deal of coughing among the others with three of them sufficiently tachypnoeic to have been taken to individual boxes as a precautionary measure. Approximately half of the remaining animals were still in the field with their dams while the other half—about 30—were in a large court. Six of them were markedly tachypnoeic ; all 16 faecal samples from this herd were negative for larvae.

The autopsy of the dead calf revealed only one adult lungworm and numerous smears of bronchial mucus failed to reveal any larvae or young adults. A widespread cuffing pneumonia was present indicating that the disease had been in progress for more than 48 hours ; the terminal symptoms were attributable to severe pulmonary oedema. This emphasises the difficulty of detecting pneumonia in the bovine in its early stages, particularly in beef breeds the respiratory rate of which is so susceptible to environmental alterations in tem-

perature and humidity. Many cases go unnoticed by owners and stockmen even when they are coughing and breathing rapidly.

It is often valuable to examine the indoor calves of the same herd for pneumonia where this condition is suspected to be the primary cause of disease.

(3) The existence of widespread epithelialisation of the alveoli. This lesion is discussed more fully under "Pathology." The point which we wish to make here is that we have seen cases in calves and adults where there was widespread epithelialisation associated with small numbers of lungworms and where the former was obviously responsible for the severity of the symptoms.

(4) Persistent pneumonia as a sequel of infection with lungworms. During the late autumn and winter we have frequently observed stirks which do not recover from a lungworm infection. They remain stunted and thin and are often pot-bellied. The respiratory rate is usually 30 to 50 per minute and auscultation usually reveals typically harsh bronchial inspirations. These persistent pneumonias associated with very few or no lungworms are often encountered in the knackeryard in early winter.

The severe cases may suddenly die or may be slaughtered as unprofitable. Autopsy reveals that the lungs are extensively damaged. The lesions are varied but extensive fibrosis of the walls of the bronchi (Fig. 1) and fibrous replacement of the alveolar tissue are common. Broncho-pneumonia and emphysema are present in varying degrees while cuffing and epithelialising lesions may play prominent parts in specific cases. In eight cases which were known to have been severely affected with parasitic bronchitis several months earlier we did not find any adult lungworms at autopsy.

If one examines these animals without being aware of a previous history of parasitic bronchitis one or more months previously, they may appear as fairly typical husk cases with no larvae in their faeces, or if it is late in the year and they are indoors, as cases of indoor pneumonia.

LESIONS ASSOCIATED WITH OUTDOOR HUSK

This account is not concerned with all of the histological changes found in husk; these are many in number and type and a separate paper will be devoted to them including both natural cases and the stage-by-stage development of the lesions in the experimental disease.

It has become progressively more apparent to us that the dyspnoea seen in husk cannot be related solely to bronchial obstruction caused by masses of worms. Several lesions develop which appear to play a great part in diminishing the ability of the lungs to effect normal gaseous exchange. It is these changes which are described below.

Cuffing Pneumonia (Figs. 2 and 3)

This has been dealt with elsewhere in this communication and its main effects may be summarised thus; a progressive lymphoid hyperplasia develops around bronchi and bronchioles and leads to (*a*) compression collapse of surrounding alveoli, (*b*) partial stenosis of bronchial and bronchiolar lumina, and (*c*) in extensive cases may lead to peribronchial fibrosis. When this lesion involves a whole lobe, almost all of the available air space is obliterated by the concomitant collapse. It may well be that lesser degrees of this lesion produce a fixed patency of bronchioles and alveolar ducts and lead to increase of pulmonary "dead space" and therefore to hyperpnoea. This latter has still to be demonstrated experimentally.

Epithelialisation of Alveoli

This term means that the pulmonary alveoli become lined with a continuous layer of epithelium; the derivation of this epithelium is still in doubt, *i.e.*, whether it is an extension of the respiratory bronchiolar epithelium into the alveoli or a proliferation of the foci of pavement epithelium considered by some workers to be normally present in the alveoli. Three different types of epithelialisation of alveoli have been found in the bovine. (1) is a low pavement type of epithelium containing eosinophilic inclusion bodies found in acute pneumonia in young calves. It has been described by one of us (Jarrett, 1954). This type is similar to that found in canine distemper, human measles and inclusion pneumonia in infants; it is possibly caused by a virus. (2) is a localised regular cuboidal epithelium found immediately adjacent to areas of peribronchial fibrosis in husk (Fig. 4). This lesion has been described in other animals in conditions producing severe bronchial trauma, *e.g.*, in human influenza and in experiments in which irritants were injected into the tracheas of animals (Watts *et al.*, 1948). (3) is the type we are mainly concerned with here. The minute lesion (Figs. 5 and 6) consists of the lining of all or most alveoli of a lobule by a sheet of cuboidal epithelium which is usually of a regular nature. In some lobules which are not wholly affected, the process is most marked at the periphery, *i.e.*, away from the bronchi. The alveoli are usually fully expanded and contain little or no inflammatory exudate. No inclusion bodies have been seen in the epithelial cells. In the cases studied to date inter-alveolar fibrosis was not a marked feature. When one considers the nature of the lesion and its obvious effect in limiting or preventing gaseous exchange across the alveolar capillary wall it is obvious that whole lobes thus affected must seriously impair the efficient working of the lung. In several cases all of the anterior lobes and the anterior strips of the diaphragmatic lobes showed complete "consolidation" by this process. The affected lobes appeared normal in volume but were of a slightly deeper colour than normal lung. They had a distinct rubbery feel.

It is noteworthy that this lesion tended to be common to all the animals autopsied in certain outbreaks. It is not known if this is a secondary pneumonia, a reaction to some specific pattern of lungworm infestation, if it occurs at a specific time in an outbreak or if it is associated with vascular or circulatory changes. We have seen this lesion in an experimental calf and have designed several experiments in an attempt to elucidate its pathogenesis. This lesion is found irrespective of the number of lungworms present in the lung at autopsy but one cannot conclude from this fact, at present, that it is independent of the degree of lungworm infestation. It often occurred in our experience when only a few lungworms were present in the bronchi but this may mean

only that the animal under consideration had shed the greater part of its lungworm load before death. In one case only has it been seen in the absence of all traces of lungworms. The case in which no trace of lungworms or larvae was found was one submitted by Captain Barker, of Hereford, and described by him as having been a case of fog fever. This showed widespread epithelialisation of alveoli. It is possible, of course, that a more extensive search would have revealed evidence of lungworm infection.

This process illustrates one way in which dyspnoea may be produced by seriously impairing the power of the lungs to effect normal gaseous exchange.

Oedema and the "Acute Heart Failure" Lesion

Varying degrees of pulmonary oedema are commonly seen in husk lungs ; this ranges from a mild localised process to a widespread process in which the lungs are sodden with fluid and difficult to lift from the carcase. In some cases, associated with oedema, cuffing pneumonia or epithelialisation, a lesion similar to the so-called "acute heart failure" or "rheumatic" lung of the human subject (Fig. 7) is found. The essential features of this lesion are (*a*) alveolar oedema, (*b*) light infiltration of the alveoli with macrophages, (*c*) congestion of the alveolar mural capillaries and (*d*) the lining of the respiratory bronchioles and alveolar ducts by pseudofibrinous hyaline membranes. Discussing the factors associated with the onset of this condition, Hadfield (1938) states, "Two factors are in operation in all these conditions. . . . Severe inspiratory dyspnoea is common to them all and this is invariably associated with a gross excess of albuminous fluid in the final airways . . . under these conditions air under tension becomes imprisoned in the alveolar ducts behind fluid exudate and the fluid around the imprisoned air is forced as a thick film or 'membrane' against the walls of the alveolar ducts during severe dyspnoea." He also states (op. cit.), "The hyaline membranes in the alveolar ducts . . . must seal off a very large number of tributary alveoli and seriously interfere with gaseous exchange."

When this lesion is prominent in husk cases it is obvious that a "vicious circle" of changes must supervene, *i.e.*, dyspnoea due to obstruction from various causes (worms, cuffing, epithelialisation) → hyaline membrane formation → interference with gaseous exchange → increased dyspnoea, and so on.

Hyaline membranes are found in the human subject in such diverse conditions as rheumatic fever, influenza, infants asphyxiated with aspirated contents of amniotic sacs, malignant nephrosclerosis with uraemia and mild cardiac failure, metastatic carcinoma of lung, Hodgkin's disease, lymphosarcomatosis, subacute bacterial endocarditis, polyarteritis nodosa and lupus erythematosus. (Epstein & Greenspan, 1941 ; Kuzma & Lustok, 1951). The essential primary factors involved have not yet been completely clarified. In husk the primary cause of the pulmonary oedema is as yet unknown ; it may not be the same in all cases.

Emphysema

This is a lesion commonly found in husk lungs (Figs. 8 and 9). The salient features are increase in volume of the affected part, absence of normal collapse when the thorax is opened, distension of the interlobular septa and crepitation on palpation. It may affect consolidated or collapsed areas when the change is largely interstitial, or "normal" areas when both alveolar and septal tissues are involved ; it may also spread to involve the hilar lymph nodes, the mediastinum and the subcutaneous tissues. The effects of this lesion in causing embarrassment to the cardio-pulmonary axis are obvious.

Bronchial Ingrowths

In some cases a peculiar lesion occurs consisting of the invasion of bronchiolar lumina by polypi which approach, in diameter, the width of the parent tube. There appears to be an overgrowth of fibroblasts in the mucosa of the bronchi and bronchioles which pushes out the epithelium at one point and then grows up the lumen so that on cross section the bronchus appears to have a central core of cellular connective tissue covered by a layer of epithelium (Figs. 10 and 11). This is yet another example of bronchial obstruction other than that caused directly by the worms.

In addition to the lesions mentioned above, bronchopneumonia and interstitial pneumonia frequently complicate the pathological picture of parasitic bronchitis. One type of pneumonia which does not seem to have been reported before is that due to the presence in the alveoli of eggs and larvae of *D. viviparus* ; these larvae are thought to be first, fourth and fifth stage but at present it is not known which of these stages is primarily responsible for the lesions. It is quite common to find areas of consolidation due to a marked "foreign body reaction" to the larvae (Fig. 12). The larvae are found lying in the alveolar lumina attached to or engulfed by large multinucleated macrophages (Fig. 13). These giant cells have a homogeneous eosinophilic cytoplasm and up to 30 to 40 coarsely reticulated nuclei. There may be so many of the latter in the cell that considerable overlapping takes place. A larva may be attacked by more than one giant cell.

In some areas where this reaction is most intense a marked necrotising process may take place (Fig. 14). The structures of the affected lobule lose their normal staining affinities and appear as a relatively amorphous bluish pink mass (in H. and E. staining) of necrotic debris with a large content of larval detritus (Fig. 15). It is possible that such a focus may act as a nidus for subsequent bacterial proliferation and so lead to an acute pneumonia. It is interesting to speculate on the possible relationship between these lesions and the phenomenon of inhibition of development described by Taylor & Michel (1954) ; we are at present conducting experiments to see if there is any relationship.

A further lesion found in the later stages of the disease and which persists afterwards, is bronchiectasis (Fig. 16) ; both the tubular and saccular types are found and constitute a common finding associated with the "wasting bovine."

This brief account of some of the lesions associated with infestation of *D. viviparus* indicates that a part of the dyspnoea shown by animals suffering from husk is due to factors other than bronchial obstruction caused by the worms themselves. This has considerable significance in several ways, *e.g.*, if one finds some drug which will kill lungworms *in situ* it does not automatically follow that the animal will make an immediate recovery ;

it explains the fact that many animals which die from "husk" show very few lungworms in their bronchi; These factors must be considered in the interpretation of faecal larval counts.

EXPERIMENTAL PARASITIC BRONCHITIS

Technique of Infection

When this work was started we had considerable difficulty in infecting calves with *D. viviparus*. The technique employed at that time was to obtain the larvae from fresh faeces using the Baermann apparatus. These larvae were then kept in a small amount of water and artificially aerated for six or more days. At the end of this time, motile larvae were counted and doses of 10,000 larvae were fed to the calves. The results were disappointing, the number of negative exceeding the number of successful infections. Various modifications such as feeding the larvae to the calves in milk, dosing by stomach tube, and giving five daily doses of 2,000 larvae, were of no advantage.

A very simple technique has now been developed which almost invariably ensures a successful infection and which involves little labour. Infected faeces are placed in beakers to a depth of 1 to 2 in. and the beakers stored in a glass tank with a glass lid. The tank contains a little water to keep the atmosphere moist. At the end of 7 to 10 days the surface of the faeces and the sides of the beaker are washed with a little water; this water is rich in very vigorous larvae. These are fed to the calves using a stomach-tube.

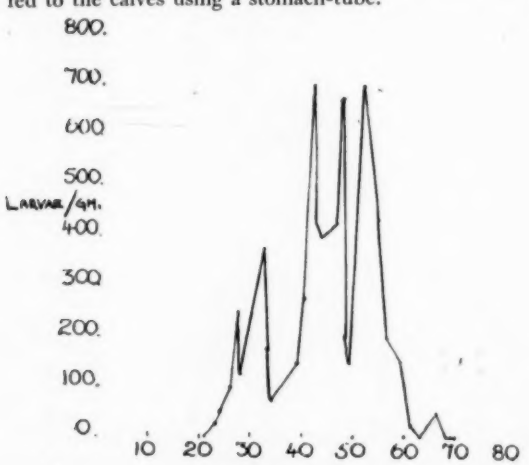

TABLE 2.—Typical larval output of an experimental calf infected at two months of age with 5,000 infective larvae of *D. viviparus*.

Size of Infecting Dose

If calves of under two months old are used, a dose of 5,000 larvae is sufficient to produce counts of several hundred larvae per gramme (*v.* Table II). In older calves, 10,000 larvae are generally used as the infecting dose. (Ayrshire calves were used throughout this work.)

Course of Infection

In successful takes gross respiratory symptoms may appear as early as 12 days after infection and auscultation may detect respiratory abnormalities as early as the fifth day after infection. These symptoms become more marked as the infection develops. A fair degree of mortality in these calves is common and two points are noteworthy in this connection :—

(*a*) Calves may frequently die about 24 days after infection, *i.e.*, just when the lungworms are becoming adult and before larvae are easily detectable in the faeces.

(*b*) Calves which are reared and infected in isolated units seem to resist the effects of an infection more easily than calves reared and infected together in a calf house of conventional pattern.

Larvae may be detected in the faeces about 23 days after infection. Their concentration in the faeces usually rises quickly and larvae will continue to be passed in easily detectable quantities for 30 to 40 days (*v.* Table II). There is some variation in the course of the larval output and this is at present being studied in conjunction with work on immunity.

Several weeks after calves have apparently ceased to pass larvae in their faeces, they may still show respiratory symptoms. If these calves are killed a few lungworms may be found in the distal parts of the diaphragmatic bronchi, although the symptoms are usually referable to persistent pneumonic lesions.

INDOOR HUSK

At most times of the year, but especially in the winter months, it is quite common to find groups of housed calves suffering from intermittent coughing. On some farms this becomes quite a serious problem and there is obvious interference with the growth rate of the calves and their utilisation of food. In such outbreaks it is common to find an occasional calf suddenly develop either an acute pneumonia or a progressive dyspnoeic condition. In our experience, most, but not all, cases of this type are due to a condition which we, in a preliminary report on the condition, have called cuffing pneumonia (Jarrett *et al.*, 1953). The basic feature of this pneumonia is the development of peribronchial cuffs of hyperplastic lymphoid tissue containing "germ centres," the extent of these cuffs and the degree of bronchial and alveolar collapse which they cause corresponding fairly closely to the clinical syndrome. At the time of our first publication we had no information about the aetiology of this disease although we were able to reproduce the condition in a serial passage by intratracheal injections of suspensions of affected lung tissue and by contract in a calf house between healthy and diseased animals. The condition appeared to have clinico-pathological specificity and was produced only by lung tissue which showed cuffing lesions.

In our subsequent work we have tried to find the aetiological factor or factors concerned and, purely as working hypotheses, we have taken into account the following factors. (1) That the disease is caused by a bacterium; (2) it is caused by a virus; (3) it is the non-specific result of chronic purulent bronchitis or broncho-pneumonia; (4) that it is a non-specific response to the continued presence of foreign material in the bronchi; (5) that it may be a sequel to one or more "specific" but clinically occult pneumonias.

These factors will be considered in reference to the experimental work briefly summarised below.

From our experience of transmitting cuffing pneumonia with lung suspensions we considered that 28 days was probably the optimal time to terminate each experiment ; animals were killed at this time after inoculation except in a few cases. The calves used were bought in a local market as cast bull calves at two to seven days old. They were kept either singly or in groups of three in isolation pens ; one to two weeks were allowed to elapse between the isolation of the calves and inoculation in order to ensure that the calves were free from respiratory disease. The calves were pail fed, observing strict sterilising and disinfecting precautions of pails and attendants' clothes respectively. Unless otherwise stated each experiment was carried out using 12 calves.

Details of Experiments

1. In order to determine if cuffing is a non-specific response to the introduction of normal homologous lung tissue, calves were inoculated intratracheally (I.T.) with 20 ml. of a 10 per cent. weight/volume suspension of normal lung suspended variously in saline, bacteriological broth, serum broth and phosphate buffer (pH 7·2). The calves did not show any clinical reaction and when killed 18 days later showed no pulmonary changes.

In addition, three calves were given I.T. injections of suspensions of pig lung in the same doses and suspending agents as above. No changes were seen at autopsy 28 days later.

2. Calves were inoculated I.T. with suspensions of lungs which showed only purulent broncho-pneumonia. The inoculated calves developed purulent broncho-pneumonia and some of them died ; those surviving until the 28th day were killed. Some of the survivors showed no lung lesions ; others had bronchitis or broncho-pneumonia. None developed cuffing pneumonia.

Another experiment, the details of which will be reported elsewhere (Dow & Jarrett) is of interest. Twelve calves were divided into six groups of two, each pair being housed in a separate pen in the same calf house. One calf of each pair was given 500,000 units of sodium penicillin one day before and daily during the experiment. The six groups of calves were then given I.T. suspensions of purulent lung in dilutions 10^{-1} to 10^{-6} respectively. With one exception (10^{-6}) the calves which received penicillin did not develop pneumonia, and with one exception (10^{-4}) those which did not receive penicillin showed purulent pneumonia 28 days after injection. The bacteria involved were a mixed flora including Clostridia, *Bacterium coli*, Lancefield negative Streptococci, *Corynebacterium pyogenes* and coagulase negative *Staphylococcus albus*. Penicillin prophylaxis as outlined above did not prevent the development of cuffing pneumonia in seven calves inoculated I.T. with " cuffing " lung suspension.

It is almost of interest to note that in some instances, but not all, the lung suspensions used were bacteriologically sterile and some " cuffing " lungs yielded no bacteria on culture. In most cases, however, it is impossible to obtain these conditions due to the presence of small quantities of pus in the bronchi.

3. Successful passages were obtained using suspensions of broncho-mediastinal lymph nodes (I.T.) from cuffing cases. Some of the nodes used were bacteriologically sterile. Control calves inoculated with normal nodes did not develop lesions.

4. Filtrate experiments. Before attempting passages with filtrates it was thought necessary to ascertain that the disease could be transmitted with dilutions of suspension since a degree of infectivity is lost on passing through a filter. It was found that passage was possible on diluting the supernatant from a centrifuged 10^{-1} dilution of lung suspension to 10^{-4}.

Similar supernatant fluids were then passed through Seitz and Gradocol filters.

Seitz EK filtrates. Five calves were inoculated with filtrates from three cuffing cases. No lesions were found in the lungs 28 days later ; no clinical abnormalities were noticed during the experiment.

Gradocol membranes. Filters of A.P.D. 0·8 u. were used. Controls were inoculated with suspensions of the same lung as was used for filtrates.

Experiment (*a*) Two calves were given filtrate (I.T.) and one suspension. All were negative.

Experiment (*b*) Two calves were given filtrate (I.T.) and were negative. The control died of intercurrent infection.

Experiment (*c*) Nine calves were given filtrate (I.T.). Six showed areas of lobular collapse at autopsy and these showed cuffing histologically ; one calf had well-developed cuffing pneumonia of the anterior lobes.

Experiment (*d*) Nine calves given filtrate (I.T.). Five showed mild pneumonia 28 days later ; this was confirmed to be cuffing pneumonia histologically.

At no time in the course of the disease did these animals show obvious pneumonic symptoms but detailed clinical examination was not carried out since strict isolation precautions were being maintained.

In addition to the experiments summarised above several others were carried out, mainly of a technical nature, *e.g.*, no advantage was found in anaesthetising the calves before injection ; intranasal instillation appeared to be inferior to intratracheal ; 20 ml. was found to be the most useful volume although 40 ml. was equally satisfactory and did not give rise to extraneous reactions. It was also found that cuffing could be transmitted by suspensions of lungworms from a field case of cuffing plus parasitic bronchitis ; however, it was found equally easy to produce the disease by bronchial fluid from the same case and no conclusions could therefore be arrived at as to the possibility of the worm carrying the infective agent.

It is difficult to draw any final conclusions as to the aetiological agent involved from the work already done. Questions of space prevent a fuller account of the experiments and the technical difficulties to be taken into account in any careful study of calf pneumonia but it appears from these experiments that cuffing is not a non-specific response although in the absence of the demonstration of a particular etiological agent it is impossible to say that only one factor is involved. It is too early to interpret the results of filtrate experiments. Several sets of circumstances may have occurred, *e.g.*, (*a*) the causative organism may be a virus, (*b*) a virus pneumonia may have been passaged and the

cuffing lesions caused by a secondary infection, (*c*) one or more of the calves may have been infected before the experiment started and natural transmission within the groups of three might have occurred.

No one bacterium has been found to be common to all cases of cuffing pneumonia ; *B. coli*, Streptococci, Staphylococci, *C. pyogenes*, *Past. septica* and a Haemophilus-like organism have been found. As has been stated above, some lungs gave no bacterial growth on culture.

Several cases of chronic pneumonia and bronchiectasis have been studied in which there has not been any cuffing reaction ; if cuffing were due to the continued presence of pus in bronchi one would expect it to have been present in these cases. In addition fatal and severe cases of cuffing have been studied in which there was little or no pus present in most parts of the affected lung tissue. The intratracheal injection of homologous normal lung, lung showing purulent pneumonia, normal lymph node tissue, and pneumonic pig lung in fairly high concentration (10 per cent. weight/volume) did not produce cuffing lesions. This also supports the view that cuffing is not a non-specific response to either foreign or cellular material in the bronchi or to prolonged toxic stimulus. It is too early to claim complete etiological specificity for any one agent and much work remains to be done on this atypical pneumonia. Extensive histological examination of many field and experimental cases leaves no room for doubt that the compression collapse of pulmonary tissue caused by the lymphoid cuffs is responsible for clinical symptoms. A full account of the clinical features of cuffing pneumonia has already been given (Jarrett *et al.*, 1953).

We have observed a similar *clinical syndrome* develop indoors in several different ways :—

1. We have seen both experimental and natural cases of indoor parasitic bronchitis occurring when animals suffering from the disease were mixed with calves which had never been to grass. This was probably due to contamination of their common bedding with larvae. We have not, as yet, found any case which can be attributed to the survival of infective larvae in hay.

2. Calves may become dyspnoeic and cough about 10 days to a fortnight after exposure to the infective stages of *D. viviparus*. If they have been brought indoors they may show typical husk symptoms while showing no larvae in their faeces. This tends to be misleading and a guarded diagnosis should be made and the possibility of fully developed parasitic bronchitis occurring a fortnight later should always be borne in mind.

3 We have also observed both experimentally and in the field cases in which husk symptoms developed after indirect contact with animals suffering from parasitic bronchitis, and it appears that this was due to the transmission of non-parasitic or secondary pneumonia from the parasitic bronchitis cases to the other calves ; this illustrates the danger of the common practice of mixing, in the same house, parasitic bronchitis cases, which have been brought in for treatment, with the young indoor stock.

4. Cases which throw off their lungworm burden may remain clinically affected and may show no larvae in their faeces and little or no lungworm infestation at autopsy. This is due to the fact that the pneumonias associated with parasitic bronchitis infestation may persist for a considerable time after the majority of parasites have been shed. These pneumonias are described in the section on pathology of husk and their significance in assessing faecal larval counts is considered in the appropriate section.

THE RELATIONSHIP BETWEEN PARASITIC BRONCHITIS AND CUFFING PNEUMONIA

In a previous report (Jarrett *et al*, 1958) we stated that we had encountered no cases of parasitic bronchitis which were not accompanied by the hyperplastic lymphocytic cuffs characteristic of cuffing pneumonia. This year, however, we have autopsied animals from several outbreaks which did not show this lesion.

Until we are able to transmit cuffing pneumonia with facility using filtrates or cultures, it is difficult to work out their exact relationship and why they should be so closely associated. In this respect we have again noted the frequency with which cuffing pneumonia as an indoor disease and severe parasitic bronchitis in the stock at pasture occur together.

On several occasions we have transmitted cuffing pneumonia to calves with suspensions of lung or lymph gland from field cases of parasitic bronchitis and this, in turn, was serially passaged through several generations of calves.

We have also reared calves in isolation, both at the veterinary hospital, and elsewhere, in premises where there have never been bovines ; these calves, when infected with lungworm larvae showed cuffing lesions. The lungs from two of these calves were injected intratracheally into two calves producing cuffing lesions in one case and no lesions in the other.

CONTROL

(*a*) Cuffing Pneumonia

The most severe outbreaks of this disease have been encountered on farms where calves of all ages from birth to several months were reared in one large calf house where it was impossible to control the spread of infection.

On those farms we have advised a system of rearing calves individually, or at the most in groups of two and three, in different sheds throughout the steading. Where this accommodation is not available the calves may be reared in the hayshed, the walls of the " pen " being bales of hay. In certain exposed areas intense cold during the winter months may render this impracticable. Individual feeding pails should be used. If possible, a different person should look after the rearing of the calves.

In our limited experience this system is giving satisfactory results. Indeed, from our experience in the field and at the veterinary hospital we are convinced that the rearing of calves in individual units is infinitely superior to the standard practice of rearing in a large communal house for calves of all ages. This latter practice is particularly suitable for the survival of

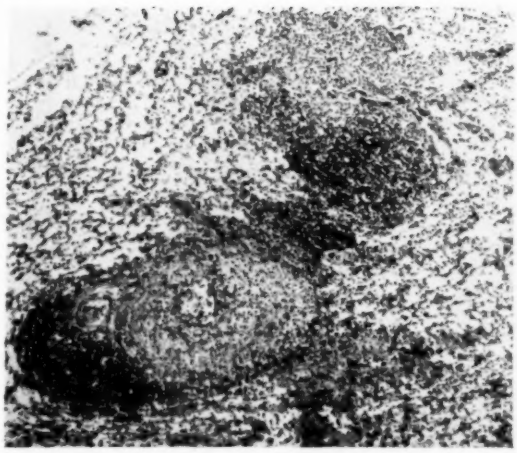

Fig. 1.—Peribronchial fibrosis in an animal which had parasitic bronchitis several months before. H. and E. × 46.

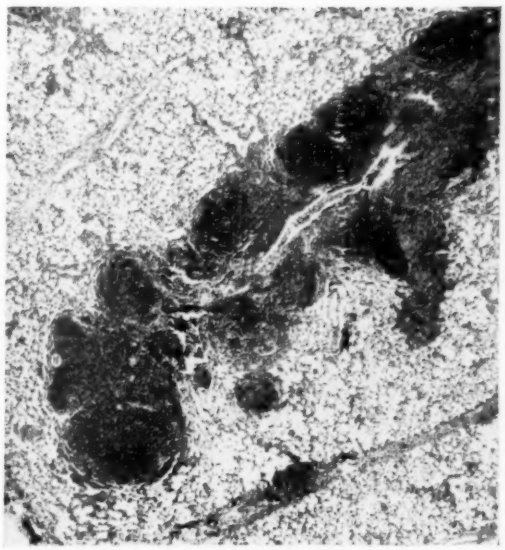

Fig. 2.—Longitudinal section of a bronchus surrounded by a continuous hyperplastic lymphoid cuff. Note formation of "germ centres' and bronchial constriction. H. and E. × 35.

Fig. 3.—Cross section of several bronchi showing lymphoid cuffs, surrounding alveolar collapse and slit-like lumina. H. and E. × 35.

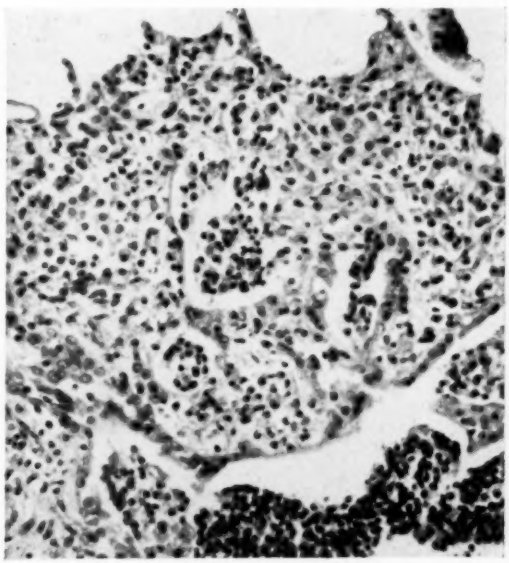

Fig. 4.—Epithelialisation of alveoli in immediate peribronchial area. H. and E. × 200.

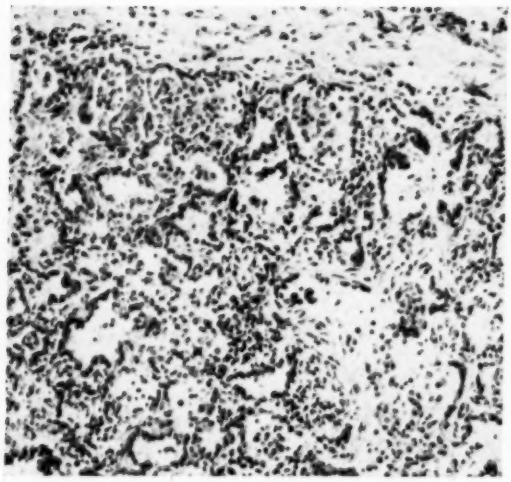

Fig. 5.—Peripheral area of a pulmonary lobule showing the uniform lining of alveoli with epithelium. H. and E. ×95.

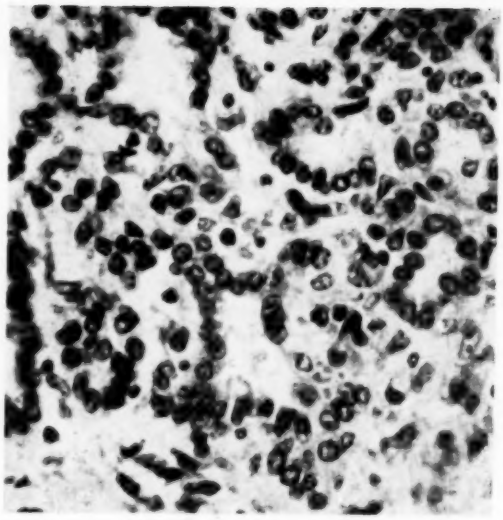

Fig. 6.—High power view of fig. 5. Relatively regular epithelium lining alveoli. H. and E. × 410.

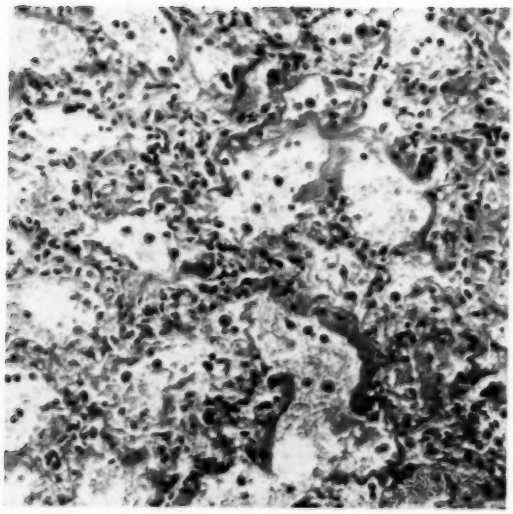

Fig. 7.—The "acute heart failure" or "rheumatic lung" lesion. Hyaline membranes are lining the alveolar ducts and, to a lesser extent, the alveoli. H. and E. × 250.

Fig. 8.—Lungs showing widespread emphysema. Note lack of collapse, glistening surface and exaggerated lobular delineation.

Fig. 9.—Close up of surface of lung (fig. 8) showing alveolar and septal emphysema and early bulla formation. × 2.

Fig. 10.—Cross section of bronchial ingrowth through point of origin in wall from an old case of parasitic bronchitis. H. and E. × 40.

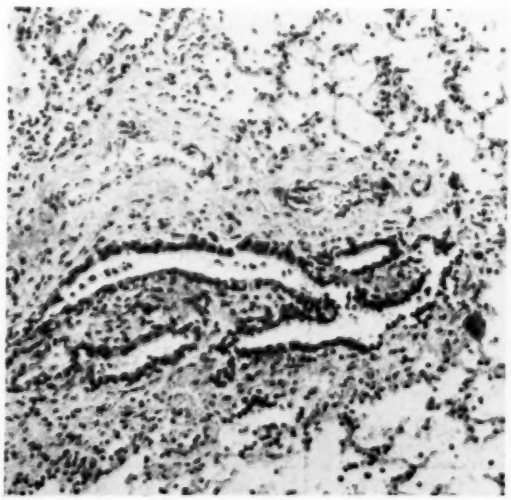

Fig. 11.—Longitudinal section of bronchiole through point of origin of a bronchial ingrowth. H. and E. × 115.

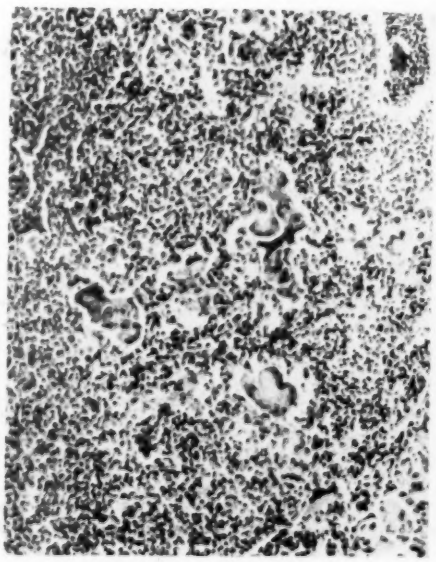

Fig. 12.—Macrophage reaction to larvae showing multinucleated giant-cells of the "foreign body" type. H. and E. × 100.

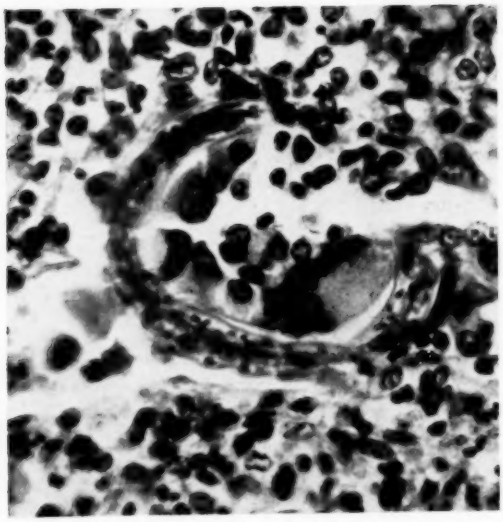

Fig. 13.—Larva (probably 4th stage) being attacked by multinucleated giant cells. H. and E. × 410.

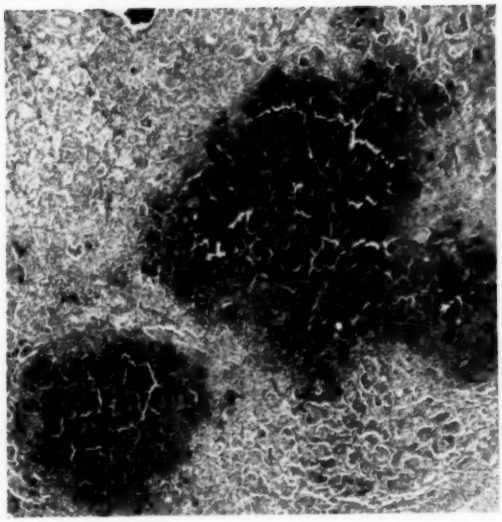

Fig. 14.—Areas of necrosis supervening on foci of intense "larval" reaction. H. and E. × 35.

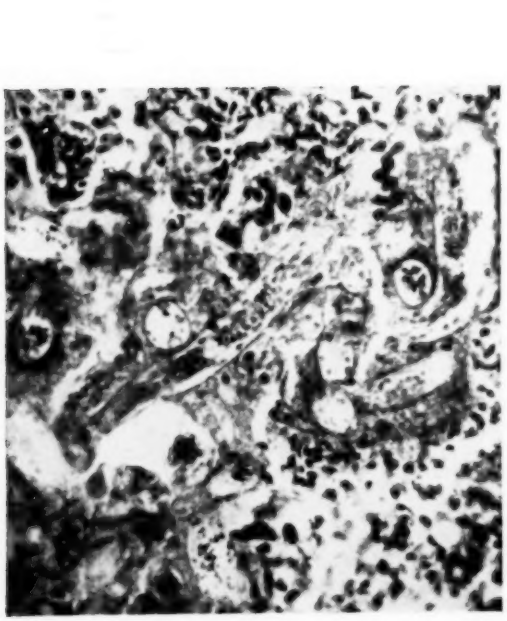

Fig. 15.—High power view of fig. 14, showing pyknotic nuclei, cell debris and larval detritus. H. and E. × 410.

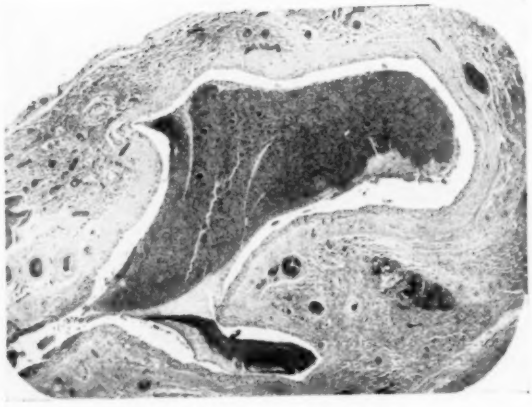

Fig. 16.—Bronchiectasis of main descending apical lobe bronchus, showing grossly dilated lumen filled with necrotic debris, absence of normal epithelium and collapse of the surrounding alveoli. H. and E. × 10.

endemic pneumonia over prolonged periods because of the close association of a wide range of age groups within the one house. A further point is that the spaciousness and ventilation of the calf house is of little importance in the control of respiratory disease as long as all age groups are confined together. During the past two years we have reared several hundred Ayrshire calves from a few days old and we have found that the individual unit greatly facilitates the production of a disease-free calf. On a commercial basis the individual unit has much to recommend it from both the financial and labour-saving points of view.

(*b*) Parasitic Bronchitis

(i) *Pasture Management*

Parasitic bronchitis differs from parasitic gastro-enteritis in that small numbers of the parasite may produce effects out of all proportion to their number and that at present it is not amenable at all to drug therapy. For these reasons control can only be effected by a strict grazing policy. This policy depends on the recognition of two facts ; namely, that the larvae survive the winter on pasture, and that animals which have had parasitic bronchitis the previous year may be a potential source of danger at least during the following year.

The essential point of the scheme is that young calves going out to grass for the first time must be reared during the whole of their first year on clean ground. By clean ground is meant first-year grass, *e.g.*, a corn crop may be undersown with grass and when the corn is taken off, sufficient acreage of the field must be fenced off and kept for the following year's calves. It should be stressed that no bovines must be allowed on this area after the corn has been taken in, nor must the field be manured. Furthermore, the calves must be confined to this field during the whole of the grazing season.

We are hoping to see the results of this technique this year in several farms which were severely affected by parasitic bronchitis during last summer, although we have already encountered instances where calves reared on ground such as is described above remained free from disease while other age groups grazing elsewhere became infected.

The scheme, however, is not without a drawback. It would appear from the frequency of cases of parasitic bronchitis in adults, that age immunity as distinct from acquired immunity is of no practical value, if indeed, it exists at all. It follows, therefore, that calves reared on clean ground during their first year, must also be grazed on clean ground during the second year. This is not difficult for farms which are wholly arable. The real difficulty arises when these lungworm-free animals join the milking herd. These young cows will probably be relatively susceptible to parasitic bronchitis and trouble may ensue unless a segregation grazing policy is used until the older cows are eliminated from the herd.

In view of the extra labour involved in carrying out segregated grazing with the cows, this policy of complete eradication is unlikely to be adopted by farmers unless parasitic bronchitis is particularly severe. Furthermore, it is of value only in self-contained herds. In spite of this we think that a scheme which provided clean grazing for cattle up to two years of age would be of great value in reducing the incidence of parasitic bronchitis. It is also of value to plough the pasture on which the outbreak has occurred as this will ensure the eradication of large numbers of larvae. This scheme is being carried out with the co-operation of the farmer on several farms which have suffered severe loss from this disease over the past two years.

(ii) *Phenothiazine*

As there is no literature on the effect of phenothiazine (PT) in parasitic bronchitis of bovines some work has been carried out on this subject.

A few preliminary experiments gave the impression that small daily doses of PT a few days before and after artificial infection reduced the number of lungworms which developed. The first experiment to test this gave disappointing results. Two groups of calves were given 5 grammes PT, one 48 hours before and one six hours before infection with 5,000 nine-day larvae. In both cases there was no difference in the number of lungworms compared to the controls.

One interesting point is that PT given orally or intratracheally will kill the first stage larvae of *D. viviparus* either in their passage through the gut or within a few days of being passed in the faeces. This effect is quite marked and in one two-year-old heifer excreting 1,000 larvae per gramme faeces, an oral dose of 15 grammes PT was sufficient to kill almost 100 per cent. of the larvae for several days. The drug appears to have no effect in reducing the faecel larval output, and this may continue to rise after treatment. Experiments are at present in progress to find if this larvicidal effect of PT is of any practical value in the control of the disease.

While we have not carried out any experiments designed specifically to find if PT has any lethal effect on adult lungworms we have observed that animals which were carrying a heavy lungworm load as indicated by very high larval counts, and which were treated with PT orally and intratracheally, showed large numbers of living lungworms at autopsy.

SUMMARY

1. This paper is an interim report on a study of husk in the bovine ; it contains a summary of a year's work on the subject.

2. The main problems of parasitic bronchitis are stated.

3. The age and seasonal incidence in the West of Scotland is described.

4. Experiments are described which show that lungworm larvae may survive for as long as 13 months on pasture ; the problem of carriers is also discussed.

5. The epidemiology and course of the disease in the field in both calves and adults is described and discussed.

6. The significance of faecal larval counts is considered.

7. Several lesions associated with outdoor husk are described ; it is considered that these lesions are often responsible for severe respiratory symptoms.

8. A brief description of experimental parasitic bronchitis in calves is given.

9. Field observations and experimental work are discussed in relation to indoor husk.

10. The relationship of cuffing pneumonia to parasitic bronchitis is considered.

11. Systems of control of cuffing pneumonia and parasitic bronchitis are suggested.

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The discussion which followed the delivery of this Paper will be published next week.

Current Literature

ABSTRACTS

Normal Variations in the Rate of Machine Milking. DODD, F. H. (1953). *J. Dairy Res.* **20.** 301–318.

The milking rate throughout milking was recorded for 141 cows of the Shorthorn, Guernsey, Friesian, and Ayrshire breeds in 326 lactations by suspending the milking machine bucket on a spring balance and recording the balance reading at half minute intervals. The results showed that the rate in the first minute, the rate of milking in the second and third minutes, (mid-milking rate), the maximum milking rate attained during milking (peak flow), and the mean rate of milking up to the time when machine stripping commenced (machine rate) were greater at the morning milking than the evening, although the total time was longer because of the much higher yield. Afternoon and morning records obtained when the yields were virtually the same showed that the rate of milking was greater in the early lactation afternoon milkings, and consequently, the total time was less. They also illustrated very clearly that between-cow differences were very much greater than within-cow variations.

An experiment to measure the lactation trends in milking rate indicated that as the morning milk yield declined the peak flow, machine rate, and machine time, (duration of milking up to the start of machine stripping) declined even when allowance was made for a stage of lactation effect. Also, as the interval since calving increased, the peak flow and machine rate decreased, and the machine time increased, independently of the yield changes. The experiments also showed clearly that the absolute and relative decline in milking rate with fall in yield was greater in the case of the fast milking cows.

It also appeared that with an increase in age there was a gradual increase both in yield and milking rate. Nevertheless the total duration of milking increased because the increase in yield was progressively greater than the increase in rate. To the farmer the most important measure of the milking rate of a cow is the total duration of milking, for slow-milking cows tend to upset the general routine of milking and increase labour costs. The total duration of milking is dependent on the amount of milk given at a milking, the rate of milking, and the yield of strippings which have to be removed by the manipulation of the milking machine. If the influence of stripping yield on the total time is given a value of one, these experiments showed that the yield is three times, and the peak flow six times as important. It was found that the percentage yield in two minutes was a useful indirect measure of milking rate. It was closely correlated with machine time and could be obtained simply without upsetting the cow and without elaborate equipment. J. O. L. K.

Pulsation and Milking Rate. CLOUGH, P. A., DODD, F. H., & HUGHES, E. W. (1953). *J. Dairy Res.* **20.** 375–380.

The object of the experiments reported was to determine the effect which variations in the frequency of pulsation had on the milking rate. The nine cows used were divided into three groups with one relatively slow, one medium, and one relatively fast milker in each. Each group received each of three experimental treatments of 20, 50, and 80 pulsations per minute. From the milk flow records obtained at the four timed milkings in each experimental period six measures of milking rate were abstracted. The mean values for these measures calculated for each cow, and for all nine cows, indicated that progressive increases in pulsation rate gave faster milking. The individual figures for each cow indicated that whereas all nine cows showed the increase in peak flow and rate in the first minute, the effect on total milking time was not apparent for three of the cows. Changes in the pulsation rate did not appear to effect total milk yield and strippings yield.

To determine whether these increases in milking rate were due to the reaction of the cows to the stimulus of the higher pulsation rates, or to changes in the mechanical conditions of the teat cups, a milking machine was constructed which milked the left side of each udder separately from the right side. This machine was used to apply pulsation at different rates simultaneously to the two sides of the udder. The five cows selected for this experiment had a wide range in milking rates. During the eight day experimental period each milking commenced with both sides of each udder subjected to 50 pulsations per minute. This was continued until the milk appeared to be flowing into the jars at a constant rate, a period which varied from 15 to 45 seconds. Following this the period of constant flow was divided into four equal intervals, the duration of which had been determined in initial trial milkings. For 38 out of 40 milkings 80 pulsations per minute gave faster milking than 50 pulsations per minute, and for 39 out of 40 milkings 20 pulsations per minute gave slower milking. Using 50 pulsations per minute as the standard, the mean relative milking rates at 20 and 80 pulsations per minute were 81.1 and 109.7 per cent. respectively. The three abnormal readings were all obtained from the same cow.

J. O. L. K.

In Parliament

SECOND READING OF THE PESTS BILL

Mr. Heathcoat-Amory, Minister of Agriculture and Food, moved the second reading of the Pests Bill, on Friday, October 22nd.

He said that to-day the only county unaffected by myxomatosis in England and Wales was the Isle of Ely, and all counties but five in Scotland were affected. To speculate about next year, it seemed likely that as we would be starting with a much wider spread than this year, the effects could be expected to be a good deal greater. On present evidence a lot of rabbits were likely to survive, and that made it important that the normal action taken to destroy rabbits should be intensified. The only way that the effects of myxomatosis could be reduced, judging by the Australian experience, was an energetic mopping-up campaign.

The humane traps advisory committee were pressing on with their efforts to find a really effective substitute for the gin trap, and half a dozen had been picked out for trial. Anyone who had designed, or who was working on a design for, a trap should lose no time in getting in touch with the committee, through his department. As an incentive the Government were proposing to make *ex gratia* monetary awards to those whose original designs led to the development of humane traps coming into general use. Details of the scheme would be made known.

Extra Powers

The Committee on Wild Animals had recommended that both on the grounds of humanity and national economy the official policy on the extermination of rabbits was correct. But if the Government were to implement that policy they needed the extra powers contained in the Bill. During committee amendments would be proposed to Clause 8 to give effect to the changes announced by his predecessor on May 27th, applying the ban on the gin trap from July 31st, 1958, but taking the power two years before to postpone the date if circumstances made it necessary. The Government were satisfied that the use of traps of some kind would be necessary in the future.

It was extraordinarily difficult to produce an alternative which would not only catch the rabbit, but which would kill it as well. At least two trapping seasons would be needed to develop and produce the new traps, and then a further period would be required for manufacture and for trying them out in practice, and to allow trappers to become proficient in their use. It was not, therefore, practicable to make the date earlier than 1958.

Mr. Somerville Hastings said it was as essential to eliminate rabbits for efficient farming as it was to eliminate tubercular cattle for efficient dairy herds. The Minister should not only designate clearance areas, but should also help farmers to get them clear, providing squads for the purpose.

Sir Roger Conant said that if a date for the abolition of the gin trap was made too early there was danger of a gap between the ending of the cruel traps and the effective operation of new and humane methods.

Mr. Clifford Kenyon said that the entire country must be designated a clearance area if the pest was to be completely exterminated. The gin trap was a diabolical instrument and was unnecessary to get rid of rabbits. There were other methods far more effective and the gin trap should be abolished from the moment that the Bill became law.

Mr. Denys Bullard said it was optimistic to suppose that the country was going to get rid of rabbits. He agreed with others who detested the gin trap, but it was right that there should be a proper interval before it was abolished.

Myxomatosis

Mr. Morley (October 21st), asked the Minister of Agriculture if he will make a statement on his policy with regard to the spread of myxomatosis among rabbits in this country.

Mr. Dudley Williams asked the Minister of Agriculture what evidence he now has that myxomatosis is being deliberately spread in the West Country.

Dr. King asked the Minister of Agriculture whether he will now introduce legislation to make the wilful spreading of myxomatosis illegal.

Mr. Amory: Myxomatosis is now present in all counties in England and Wales but one, and in 28 counties in Scotland. I have read reports about the deliberate spreading of this disease by individuals, but I have no direct evidence of this having been done in particular cases.

The Myxomatosis Advisory Committee considered at a meeting yesterday whether legislation should be introduced to make the wilful spreading of myxomatosis a punishable offence. While reaffirming their view that no attempt should be made to assist the spread of myxomatosis or to introduce it into unaffected areas of the country, the Committee unanimously recommended that, having regard to the course the disease had taken and might be expected to take in the future, no good purpose would be achieved at present by making it an offence for any person to take steps to spread the disease.

Major Beamish asked the Minister of Agriculture if he will make a statement about the extent of myxomatosis among tame and wild rabbits or other animals; and whether sufficient supplies of vaccine are available to meet the demand.

Mr. Amory: Very few cases of myxomatosis among tame rabbits have so far been brought to the notice of my Department. Regarding wild rabbits, myxomatosis is now present in all counties in England and Wales but one, and in 28 counties in Scotland. No animals other than the rabbit are known to have been affected with myxomatosis in this country. I understand that the vaccine available commercially is equivalent to more than 12 months' supply at the present rate of demand.

Protection of Attested Cattle Purchasers

Mr. Hayman (October 21st), asked the Minister of Agriculture what steps he proposes to take to pro-

tect more fully purchasers of attested cattle, in view of a defect in the present methods revealed in correspondence which has passed between the Parliamentary Secretary and the hon. Member for Falmouth and Camborne.

Mr. Amory: I am considering whether any changes can usefully be made in the administration of the Attested Herds Scheme. The main responsibility must, however, rest with the purchasers to see that they obtain attested cattle only from reputable sources and that all animals purchased are accompanied by the official permit identifying the animal. I am taking steps to impress on farmers the importance of this.

Mr. Hayman: While thanking the Minister for that reply, may I draw his attention to the fact that the correspondence to which I have referred in my Question indicates possible loopholes for sharp practice by cattle dealers? Will he, therefore, do all he can to protect the buyers of attested cattle?

Mr. Amory: I realise that there are possible loopholes for sharp practice. I am looking into this, and I am grateful to the hon. Gentleman for having raised the matter with me.

Ritual Slaughter of Animals

Lieut.-Colonel Bromley-Davenport (October 20th), asked the Minister of Food when he proposes to make new regulations under Section 2 of the Slaughter of Animals (Amendment) Act for the purpose of securing humane conditions and practices in connection with the slaughter of animals in slaughterhouses and knackers' yards; whether he will draft these regulations so as to deal with the conditions under which the ritual slaughter of animals for human consumption in such places is still to be permitted; and what procedure will be adopted for consultation about such new regulations with the experts and interested organisations concerned.

Mr. Amory: Proposals for regulations including the ritual slaughter of animals were circulated for comment to the organisations concerned on September 30th and it is hoped to make regulations by the end of November.

ANIMAL HEALTH SERVICES REPORT, 1953

This Report was published on October 20th. As in previous years, accounts are given of the outbreaks of contagious diseases of animals during the year, the progress towards the eradication of bovine tuberculosis and other diseases and the preventive measures against the introduction and spread of disease from abroad. During 1953 there was a greatly reduced incidence of foot-and-mouth disease and anthrax, but sharp increases in the numbers of outbreaks of swine fever and fowl pest. This was the first year in which there was no outbreak of sheep scab and the opportunity has been taken to review the progress of the control of the disease up to what is hoped to be its final eradication.

Other sections of the Report deal with the measures for the protection of animals in transit by sea, rail and road, animal health questions in connection with the export trade, the research work carried out by the Ministry's Veterinary Laboratories and Investigation Service, and artificial insemination. The Report is available, price 3s., from the Stationery Office. It will be reviewed in more detail in a subsequent issue of this journal.

ROYAL COUNTIES DINNER-DANCE

The Royal Counties Division held a most successful dinner-dance at Skindles Hotel, Maidenhead, on Friday, October 15th, which was attended by approximately 100 people.

This is the first function of its kind held by the Division since the last war and it is hoped that it will become an annual event. Unfortunately the President of the Division, Major J. M. Smith, O.B.E., was unable to be present owing to the illness of his wife.

(*Continued from page 664*)

The untimely death of Dr. Donatien in August, 1954, robbed the conference of the opportunity of hearing this famous scientist present the paper on Piroplasmoses, a subject which has been particularly linked with the Institut Pasteur of Algeria, where he worked for so many years. He was able to finish his paper before his death, and it was introduced by Dr. Poul. As a mark of respect the conference stood silently for a few moments at the beginning of the meeting on Piroplasmoses.

Dr. Louw, of South Africa, was unable to be present to open the discussion on Mineral Deficiencies, and his paper was introduced by Mr. Anderson.

The official discussions were well interspersed with social functions, including a Reception given by the Governor-General of Algeria in the magnificent Summer Palace. Here the delegates entered between the ranks of a Guard of Honour wearing traditional Arabian uniforms, and bearing swords. Excursions were also arranged to the Casbah of Algiers, and visits were paid also to the experimental farm of Ouled-Mendil (Institut Pasteur) and to the Institut Agricole d'Algérie.

The conference in general was a very great success, and Dr. Jore d'Arces and his Organising Committee are to be congratulated on the wonderful way in which they arranged the meetings. Although the official part of the conference finished on Saturday, October 23rd, many of those present were staying on for excursions to various parts of Algeria which had been arranged for the week following.

The conference finished with a series of resolutions, which we hope to report fully in a later number of THE VETERINARY RECORD. It was generally agreed that the conference had served a useful purpose, and a permanent Committee of 25 members was set up to consider future arrangements for further Pan-African conferences of this kind.

H. E. H.

News and Comment

UNIVERSITY OF EDINBURGH—CHAIR OF PATHOLOGY

Professor G. L. Montgomery, T.D., PH.D., M.D., F.R.F.P.S., has taken up his appointment to the Chair of Pathology at Edinburgh University, and delivered his inaugural address entitled "Pathology—the Science of Disease," on October 6th. Professor Montgomery previously held the St. Mungo-Notman Chair of Pathology of Glasgow University. He succeeds Professor A. M. Drennan, M.D., F.R.C.P.E., who has retired.

DR. W. IAN M. McINTYRE

At a Graduation Ceremony held at Edinburgh University on Saturday, October 23rd, 1954, the Degree of PH.D. was conferred upon W. Ian M. McIntyre. *Thesis*—"Nephritis in the Dog associated with Leptospira Canicola Infection." Dr. McIntyre is senior lecturer in the Clinical Department of the University of Glasgow Veterinary School.

COMING EVENTS

Nov. 2nd.—General Meeting of the Scottish Metropolitan Division, B.V.A., in the Royal (Dick) School of Veterinary Studies, Edinburgh, 2.30 p.m.

Nov. 3rd.—Annual General Meeting of the West of Scotland Division, B.V.A., in the University of Glasgow Veterinary School, 83, Buccleuch Street, Glasgow, 2.30 p.m.

Nov. 4th.—General Meeting of the Central Division, B.V.A., at the Royal Veterinary College, Camden Town, N.W.1, 5.30 p.m.

Nov. 6th.—General Meeting of the Society of Women Veterinary Surgeons, B.V.A., in the Veterinary School, Park Row, Bristol, 2.30 p.m.

Nov. 10th.—Meeting of the Southern Counties Division, B.V.A., at the Red Lion Hotel, Salisbury, 6.30 p.m.

Nov. 11th.—Ordinary General Meeting of the South-Eastern Division, B.V.A., at the Princes Hall, Royal Star Hotel, Maidstone, 2.30 p.m.

Nov. 16th & 17th.—B.V.A. Conference on the Feeding of Farm Livestock for Health and Production in the South Hall, Victoria Halls, Bloomsbury Square, London, W.C.1. Admission by ticket only. Please apply not later than November 8th, on form published with issue of October 9th.

Nov. 18th.—Meeting of the Essex Veterinary Society at the Essex Institute of Agriculture, Writtle, Nr. Chelmsford, Essex, 7.30 p.m.

Nov. 18th.—Victoria Veterinary Benevolent Fund, County of Ayr Ladies Guild Dance, in the Western House, Ayr, 8 p.m. *(For details see page 680).*

Dec. 3rd.—Annual Dinner and Dance of the North Wales Division, B.V.A., in the Imperial Hotel, Llandudno.

NOVEMBER MEETINGS OF COMMITTEES AND COUNCIL OF THE B.V.A.

AT No. 7, MANSFIELD STREET, W.1.

Wednesday, November 3rd.—
11 a.m. Veterinary State Medicine Committee.
2.30 p.m. Parliamentary & Public Relations Committee.
4 p.m. Home Appointments Committee.

Thursday, November 4th.—
10.30 a.m. Organising Committee.
12 noon Finance Sub-committee.
2 p.m. General Purposes & Finance Committee.

AT CONNAUGHT ROOMS, GREAT QUEEN STREET, W.C.1.

Friday, November 5th.—
10.30 a.m. Council Meeting.

R.C.V.S. EXAMINATIONS

Friday, November 5th, 1954. Pharmacology etc. Examination (Revised Syllabus). Written (London and Dublin).

Monday, November 8th, 1954. Pharmacology examinations (Practical and Orals commence).

Wednesday, November 10th, 1954. Animal Management Examination tour commences.

Thursday, December 9th, 1954. M.R.C.V.S. Written Examinations.

Friday, December 10th, 1954. M.R.C.V.S. Written Examinations.

Monday, December 13th, 1954. M.R.C.V.S. Practical and Orals commence (Panels "A" and "B").

PERSONAL

Births

CALLEAR.—On October 14th, 1954, at Georgetown, British Guiana, to Norah, wife of John Callear, M.R.C.V.S., a brother for Diana—Richard Marcus Ratcliffe.

CAMERON.—On October 20th, 1954, at The Royal United Hospital, Bath, to Norah (*née* Hill), wife of John Cameron, B.SC., M.R.C.V.S., D.V.S.M., 49, Englishcombe Lane, Bath, a son.

CROOKS.—On October 11th, 1954, to Heather (*née* Broumpton), wife of John Crooks, M.R.C.V.S., 28, Norwood, Beverley, Yorks, a daughter.

HARBOURNE.—On October 11th, 1954, at St. Mary's Hospital, Leeds, to Ann (*née* Lawson), wife of J. F. Harbourne, B.V.SC., M.R.C.V.S., of 43, The Drive, Leeds, 16, a daughter—Elizabeth Mary.

Marriage

CHAUDHARY—KHAN.—The marriage is announced between Saeed Asghar, B.V.SC., M.R.C.V.S., second son of Nadir Ali Chaudhary and Begum Nadir Ali, and Zubida Khatoon Alam Khan, eldest daughter of Khan M. Z. Alam Khan, Barrister-at-Law, Under Secretary, Ministry of Agriculture to the Government of Punjab, on October 27th, 1954, at "Phalwari" 73, Samanabad, Lahore, Pakistan.

VICTORIA VETERINARY BENEVOLENT FUND

The Annual Dance of the County of Ayr Ladies Guild will be held this year in the Western House, Ayr, on Thursday, November 18th. Tickets are available from Mrs. A. L. Wilson, 31, Ashgrove Street, Ayr.

FEEDING OF FARM ANIMALS FOR HEALTH AND PRODUCTION

As our readers know, the Conference is open to all members of the B.V.A., but a percentage of places has been allotted also for distribution to the animal feeding stuffs trade. Other interested organisations have been invited to send a few representatives each. Admission is by ticket only. Will members *please apply for these immediately*, on the slip to be found at the bottom of the programme published with the October 9th issue of THE VETERINARY RECORD. If this has been mislaid, members are asked to write without delay to the General Secretary stating whether they require tickets for one or both days, and whether they wish luncheon arrangements made on one or both days. The closing date for such applications is November 8th. After this a further allocation of tickets will be made to the animal feedings stuffs trade and there may then be insufficient available for use by B.V.A. members.

ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax:

Cumberland. Beech House, Cumwhinton, Carlisle (Oct. 21).

Derbys. Manor Farm, Barrow-on-Trent (Oct. 18).

Leics. Fosse Fields Farm, Broughton Lane, Stoney Stanton (Oct. 18).

Staffs. Norton Farm, Watling Street, Norton Canes, Nr. Cannock (Oct. 18).

Fowl Pest:

Kent. Posingford Farm, Hartfield, Tunbridge Wells (Oct. 21).

Middx. Moorcroft Farm, Hillingdon, Uxbridge (Oct. 21).

Norfolk. White House, Yaxham, Dereham; Thorpe Farm, Shipdham, Thetford (Oct. 19).

Notts. The Rookery, Barnstone (Oct. 16).

Swine Fever:

Bucks. Piggeries at Finch Lane, London Road, Amersham (Oct. 21).

Cambs. Main Street, Coveney, Ely (Oct. 18).

Ches. Heath Farm, Heath Lane, Childer Thornton, Wirral (Oct. 20).

Essex. The Piggeries, Leaden Roding, Great Dunmow (Oct. 19); "Glencote," Main Road, Great Oakley, Harwich (Oct. 20).

Glam. Rose Cottage Piggery, Harbour Road, Grangetown, Cardiff (Oct. 21).

Hunts. Margetts Farm, Offord Road, Buckden (Oct. 18).

Kent. Wilsley Farm, Cranbrook (Oct. 19).

Lancs. Carr House Farm, Broad Lane, Grappenhall, Warrington (Oct. 18); Carver Fold Farm, Higher Walton, Preston (Oct. 20); Thurston Fold Farm, Whitefield (Oct. 21).

Lincs. Vale Farm, Barrowby, Grantham; Stone House Farm, Burton Road, Lincoln (Oct. 18); Westgate, Ruskington, Sleaford (Oct. 19).

Monmouth. The Bungalow, Malpas Road, Caerleon, Newport (Oct. 18).

Staffs. Oak House, Denstone, Uttoxeter (Oct. 23).

Suffolk. "The Jollies," Jolly Road, Mildenhall (Oct. 22).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

WILLIAM HUNTING

Sir,—It is somewhat sad that no one has yet supported Major Hancock *re* the removal of the memorial of William Hunting from the front of his beloved RECORD. In eradicating glanders from the thousands of horses in London stables, without the aid of the much simpler test applied to-day, he did incalculable service both to horse and man. His book on glanders (not to mention that on shoeing) might well be studied to-day, the illustrations of glanders lesions are extraordinarily true and the horse is still with us, if in much fewer numbers.

I have always understood that William Hunting and THE RECORD were synonymous, and I personally consider it would be a very gracious act to restore the medallion; it is a marvellous likeness of, one may truly say, a famous man in the profession in his day.

Yours faithfully,

WILLIAM TRIGGER,

Newcastle, Staffs.

October 19th, 1954.

Sir,—May I send a line in support of Major Hancock's plea for the features of William Hunting to be restored to the front page of THE VETERINARY RECORD. To see THE RECORD without its founder's

portrait is like looking at an advertisement for Dunlop tyres without the features of the late inventor of the tyre—another veterinary surgeon.

Yours faithfully,

THOMAS LISHMAN,

23, Queen's Gate Gardens,
London, S.W.7.

October 13th, 1954.

A comparatively large correspondence suggests that readers who favour the recent changes in THE RECORD greatly outnumber those who regret the omission of William Hunting's portrait from the cover.—EDITOR.

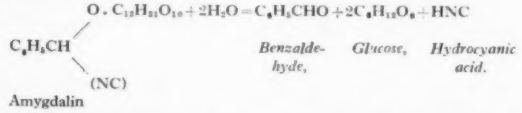
PLUMSTONES IN PIG SWILL

Sir,—In the last weeks I have had two cases in my practice where farmers' bacon pigs were condemned at the meat inspection for almond taste in the carcases. The almond taste is very distinct whether the pork is fresh, salted, boiled or fried. The pork is not dangerous to eat, but after a meal you may have almond taste in your mouth for a couple of days.

The pigs were fed with swill containing lots of plums. They also got maize and home grown foodstuffs such as oats and wheat. By examination of the foodstuffs there was nothing found but plumstones which could cause the almond taste.

This autumn there has been an abnormal quantity of plums in Norway, and those condemned have been fed to pigs. There have also been a lot of plumstones in the swill. Some pigs don't eat the stones, but mostly they crush them and seem to like the peculiar taste from the kernel.

The plum tree belongs to the family Amygdalacea as the apricot, peach, cherry and almond tree do. The kernel contains the glucoside Amygdalin which may be split up by the enzyme Emulsin or dilute acid into hydrocyanic acid, benzaldehyde and glucose.



When the pigs crush the stones, the benzaldehyde and the hydrocyanic acid will be absorbed in all the pigs' tissues and cause almond taste in the meat. We know that the carcase of an animal dead from poisoning by hydrocyanic acid has an obnoxious smell of almond from all the body tissues.

Pigs fed abundantly on plums and slaughtered a fortnight after the plums are withdrawn from the food, have no almond taste in the carcases.

There would seem to be reason to utter a caution against throwing plumstones in the swill, or feeding bacon pigs with plums. As far as I know this is the first report where plums are the cause of almond taste in pork.

Yours faithfully,

JOHANNES AUSTVOLL,

Stavanger, Norway.

October 12th, 1954.

CANINE DISTEMPER

Sir,—In "A Survey of Canine Distemper," published in THE VETERINARY RECORD of October 16th, the authors write: "It appears quite possible that hardening of the pads was a characteristic of some cases of distemper long before attention was drawn to the fact (Innes, 1949; Fankhauser, 1951; Pugh, 1948)."

Whilst practising in Cornwall during the spring of 1914, I attended an outbreak of distemper in a kennel of black cocker spaniels. Out of approximately 20 dogs, one adult and four puppies died and several affected animals made slow but eventual recoveries. A number of the inmates showed no symptoms, probably having acquired an immunity previously to this outbreak.

All the survivors developed hyperkeratosis of noses and pads, to such a degree in the latter case that thick layers peeled off the soles of the pads and some dogs actually developed lameness as a consequence. The blame for this state of affairs was attached to a young kennelmaid who was accused (in spite of her denials) of having scrubbed the floors with too high a concentration of a well-known brand of a coal-tar disinfectant.

This explanation might have remained satisfactory to everybody concerned (apart from the kennelmaid), had not the infection been carried to several neighbouring kennels, the inmates of which developed precisely similar symptoms, including those relating to the noses and feet. The sole difference between the first kennel and the latter was that the fresh outbreaks were characterised by a complete absence of both kennelmaids and disinfectant.

Soon after this, more pressing matters distracted our attention from distemper, but some years later when breeding recommenced in the same locality, our patients had reverted to the old-fashioned type of the disease with thick purulent ocular and nasal discharges and a satisfactory recovery rate.

In 1936, the character of the disease again commenced to change and cases began to appear which were ushered in by a primary encephalitis; the majority of dogs affected died or were destroyed within a very few days after continuous convulsions. Some months later, the course of the disease became more protracted and less acute (most of the highly susceptible animals being by this time dead) and those dogs which outlasted the first fortnight began to develop definite signs of pedal hyperkeratosis, to such an extent that when brought into a room, the floor of which was covered with linoleum, the pads set up a rattle which was almost clinically diagnostic.

One fox-terrier patient which recovered and was afterwards successfully exhibited was christened "Toedancer," the name having been suggested by the noise made by her feet whenever she was led into my waiting room.

An early clinical symptom, seen in practically all distemper cases at this time, was a marked enlargement of the prescapular glands, and in 1942 I observed this same symptom in dogs exhibiting

signs of acute encephalitis in the Nottingham area. These appear to have been the originators of the "hard pad" which has persisted up to this time in that neighbourhood.

Yours faithfully,

R. H. SMYTHE,

6, Peveril Drive,
The Park, Nottingham.

October 16th, 1954.

SOME REFLECTIONS ON THE MASTITIS PROBLEM

Sir,—In an interesting paper under the above title, Mr. D. L. Hughes reports results of bacteriological examinations of every case of mastitis seen prior to treatment in one veterinary practice. He says: "Do these results reflect conditions in other parts of the country? The question is not easily answered, for to the best of my knowledge results of similar surveys of every clinical case prior to treatment have not been published." This is surprising as a paper describing a survey of this type, in which detailed clinical records were kept, was published by Berger & Francis (1951). Arthur (1947) published the results of examining all cases of mastitis in five herds, containing a total of 500 cows over a five-year period. Berger visited 83 herds, containing 2,397 cows (average 28.8 cows per herd), and 7·3 per cent. of these cows were treated for mastitis per annum, which compares with Hughes's figure of 14 per cent. and Arthur's figure of 15.8 per cent. The percentage of *Str. agalactiae* isolated by Berger & Francis was much higher than by Hughes, or by Arthur. But by 1950, three years after our detailed survey was completed, the percentage of *Str. agalactiae* isolated in the practice had dropped to 10 per cent., and this infection had been eradicated from eight herds.

Hughes rightly states that failure of penicillin to cure an infection may be due to the fact that it does not reach the bacteria, and emphasises the importance of studying new ways of giving drugs. He does not, however, mention the work of McFarlane & Rennie (1950), who observed the fate of vegetable black injected into udders. They concluded that "treatment of the dry cow would appear likely . . . to be a highly efficacious method": this was the method found most effective by Berger & Francis.

Hughes discusses the question of age incidence. We observed only eight cases of mastitis associated with *Str. agalactiae* during the first lactation, but 52 associated with "other organisms." In the fourth lactation the figures were 21 and 35 respectively.

Both Arthur's survey and our own showed that the total incidence of mastitis was lowest in the second quarter of the year and highest during the third. A more detailed analysis of our own findings showed that the seasonal incidence was different in dry and lactating cows, and varied with different organisms.

The incidence of mastitis was four times greater during the first month after calving, and during the preceding three months of the dry period, than it was, on the average, during the next four months. Arthur (1947), and Neave *et al.* (1950) also noted the very high incidence of mastitis during the dry period. Evidence of injury was seen in only 19 cases of mastitis, and *Str. dysgalactiae* was isolated from 12 of these, whereas it was isolated from only 13 per cent. of all cases.

The percentage of cows, observed by Berger and by Arthur, showing systemic symptoms, were very similar and their findings confirm those of Minett, Stableforth & Edwards (1932). *C. pyogenes* produced the most severe reaction in both lactating and dry cows. *Str. zooepidemicus* produced equally severe reactions in lactating cows, followed by *Str. faecalis* and *Str. dysgalactiae*. One striking fact was that "sterile" mastitis was more severe than that associated with *Str. agalactiae*. In our survey there was very little correlation between the numbers of any given organism found in the secretion and the severity of clinical symptoms.

It will be seen that the type of bacteria, season, and stages of lactation, all have interrelated effects on the incidence of mastitis, and more complete studies may throw further light on factors which influence susceptibility to mastitis.

Yours faithfully,

JOHN FRANCIS,

The Veterinary School,
University of Queensland,
Brisbane.

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ADVERTISER'S ANNOUNCEMENT

Bayer Products Ltd. announce the removal of their administrative headquarters to more extensive premises on October 30th. From this date the new address, to which all communications should be sent, will be:—

Neville House, Eden Street, Kingston-upon-Thames, Surrey.

The addresses of the Company's factories at West Molesey and Byfleet, and of the Bayer Veterinary Biological Institute at Newmarket, will remain as at present, whilst a small branch office will be maintained at:—

Africa House, Kingsway, London, W.C.2.

The associated export company, Winthrop Products Ltd., also moves to the new headquarters at Kingston. Neither company has any connection with Farbenfabriken Bayer of Germany.